

## ACRYLONITRILE

Acrylonitrile is a federal hazardous air pollutant and was identified as a toxic air contaminant in April 1993 under AB 2728.

CAS Registry Number: 107-13-1

CH<sub>2</sub>CHCN

Molecular Formula: C<sub>3</sub>H<sub>3</sub>N

Acrylonitrile is a colorless, volatile liquid that is soluble in water and most common organic solvents such as acetone, benzene, carbon tetrachloride, ethyl acetate, and toluene. It has a fairly high vapor pressure, and if spilled would volatilize rapidly (Howard, 1990). Acrylonitrile is a reactive chemical that polymerizes spontaneously and can explode when exposed to flame (NTP, 1991).

### Physical Properties of Acrylonitrile

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Synonyms: 2-propenenitrile; vinyl cyanide; cyanoethylene; Acritet; Fumigrain; Ventox

Molecular Weight:	53.06
Boiling Point:	77.3 °C
Melting Point:	-82 °C
Flash Point:	0 °C (32 °F) open cup
Vapor Pressure:	107.8 mm Hg at 25 °C
Density/Specific Gravity:	0.8004 at 25/4 °C (water = 1)
Vapor Density:	1.9 (air = 1)
Log Octanol/Water Partition Coefficient:	0.25
Water Solubility:	75,000 mg/L at 25 °C
Henry's Law Constant:	1.10 x 10 <sup>-4</sup> atm-m <sup>3</sup> /mole at 25 °C
Conversion Factor:	1 ppm = 2.17 mg/m <sup>3</sup>

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(Howard, 1990; HSDB, 1991; Merck, 1983)

## SOURCES AND EMISSIONS

### A. Sources

Acrylonitrile is used in the production of acrylic fibers and other chemicals and resins. It can also be found in auto exhaust, cigarette smoke, and releases from the manufacture of acrylic fibers and plastics (Howard, 1990). The primary stationary sources that have reported emissions of acrylonitrile in California are synthetics, paint, and furniture and fixtures manufacturing facilities

(ARB, 1997b).

## B. Emissions

The total emissions of acrylonitrile from stationary sources in California are estimated to be at least 2,700 pounds per year, based on data reported under the Air Toxics “Hot Spots” Program (AB 2588) (ARB, 1997b).

## C. Natural Occurrence

Acrylonitrile is not known to occur as a natural product (Howard, 1990).

# AMBIENT CONCENTRATIONS

No Air Resources Board data exist for ambient measurements of acrylonitrile. However, the United States Environmental Protection Agency (U.S. EPA) has compiled information from four U.S. urban locations that reported a mean concentration of 0.66 micrograms per cubic meter ( $\mu\text{g}/\text{m}^3$ ) or 0.30 parts per billion in 1981 (U.S. EPA, 1993a).

# INDOOR SOURCES AND CONCENTRATIONS

Acrylonitrile is used in manufacturing acrylic fabrics and carpets; however, the possible release of the monomer from these products has not been quantified (Hodgson and Wooley, 1991). Acrylonitrile emissions from cigarette brands popular in California were 99 micrograms per cigarette (Daisey et al., 1994).

Data on indoor concentrations of acrylonitrile are extremely limited. During June of 1990, 125 households in Woodland, California, were monitored for a variety of toxic air contaminants. Acrylonitrile was present at measurable concentrations in only 4 of 47 samples. The mean of those 4 samples was  $9.1 \mu\text{g}/\text{m}^3$ , and the maximum measured concentration was  $27 \mu\text{g}/\text{m}^3$ . The detection limit for acrylonitrile was  $2.1 \mu\text{g}/\text{m}^3$ . These results suggest that few homes have measurable levels of acrylonitrile, but those that do may have a specific source for it since those homes have relatively high concentrations (Sheldon et al, 1992).

# ATMOSPHERIC PERSISTENCE

The dominant tropospheric chemical loss process for acrylonitrile is by gas phase reaction with hydroxyl radicals. The calculated half-life of acrylonitrile due to gas-phase reaction with the hydroxyl radical is estimated to be about 2.4 days. The products of the hydroxyl radical reaction in the presence of nitrogen oxides are formaldehyde and formyl cyanide (Atkinson, 1995).

# AB 2588 RISK ASSESSMENT INFORMATION

The Office of Environmental Health Hazard Assessment reviews risk assessments submitted under the Air Toxics “Hot Spots” Program (AB 2588). Of the risk assessments reviewed as of April 1996, acrylonitrile was the major contributor to the overall cancer risk in 2 of the approximately 550 risk assessments reporting a total cancer risk equal to or greater than 1 in 1 million and contributed to the total cancer risk in 9 of these risk assessments. Acrylonitrile also was the major contributor to the overall cancer risk in 1 of the approximately 130 risk assessments reporting a total cancer risk equal to or greater than 10 in 1 million, and contributed to the total cancer risk in 3 of these risk assessments (OEHHA, 1996a).

For the non-cancer health effects, acrylonitrile contributed to the total hazard index in 1 of the approximately 89 risk assessments reporting a total chronic hazard index greater than 1 (OEHHA, 1996b).

## HEALTH EFFECTS

Probable routes of human exposure to acrylonitrile are inhalation and dermal contact.

Non-Cancer: Acrylonitrile is a central nervous system depressant and a respiratory irritant. It is metabolized to cyanide. Symptoms include headache, dizziness, nausea, feelings of apprehension and nervous irritability, muscle weakness, cyanosis, and convulsions. In one study, workers chronically overexposed frequently reported headaches, nausea, fatigue, and weakness (U.S. EPA, 1994a).

A chronic non-cancer Reference Exposure Level (REL) of  $2 \mu\text{g}/\text{m}^3$  is listed for acrylonitrile in the California Air Pollution Control Officers Association Air Toxics “Hot Spots” Program, Revised 1992 Risk Assessment Guidelines. The toxicological endpoints considered for chronic toxicity are the respiratory system and the skin (CAPCOA, 1993). The U.S. EPA has established a Reference Concentration (RfC) for acrylonitrile of  $2 \mu\text{g}/\text{m}^3$  based on degeneration and inflammation of nasal respiratory epithelium in rats. The U.S. EPA estimates that inhalation of this concentration or less, over a lifetime, would not likely result in the occurrence of chronic, non-cancer effects. The oral Reference Dose (RfD) is under review (U.S. EPA, 1994a).

No information is available on adverse reproductive or developmental effects in humans from overexposure to acrylonitrile. In rat studies, inhalation exposure has been reported to cause fetal malformations, and in mice, orally exposed, degenerative changes in testicular tubules and decreased sperm count were observed (U.S. EPA, 1994a).

Cancer: There is limited evidence of increased lung cancer in workers chronically exposed to acrylonitrile. The U.S. EPA has classified acrylonitrile in Group B1: Probable human carcinogen. The U.S. EPA has calculated a cancer inhalation unit risk estimate of  $6.8 \times 10^{-5}$  (microgram per cubic meter)<sup>-1</sup>. The U.S. EPA estimates that if an individual were to breathe air containing acrylonitrile at  $0.01 \mu\text{g}/\text{m}^3$  over an entire lifetime, that person would theoretically have no more than a 1 in 1 million increased chance of developing cancer as a direct

result of breathing air containing this chemical (U.S. EPA, 1994a). The International Agency for Research on Cancer has classified acrylonitrile in Group 2A: Probable human carcinogen, based on sufficient evidence in animals and limited evidence in humans (IARC, 1987a).

The State of California has determined under Proposition 65 that acrylonitrile is a carcinogen (CCR, 1996). The inhalation potency factor that has been used as a basis for regulatory action in California is  $2.9 \times 10^{-4}$  (microgram per cubic meter)<sup>-1</sup> (OEHHA, 1994). In other words, the potential excess cancer risk for a person exposed over a lifetime to  $1 \mu\text{g}/\text{m}^3$  of acrylonitrile is estimated to be no greater than 290 in 1 million. The oral potency factor that has been used as a basis for regulatory action in California is 1.0 (milligram per kilogram per day)<sup>-1</sup> (OEHHA, 1994).